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CALCULATION OF PULSE WAVE PARAMETERS WITH ACCOUNT OF BLOOD VESSEL DEFORMATION

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Abstract. Self-regulation mechanisms of arterial blood flow making provision for optimum functioning cardiovascular system are discussed. Pulse wave characteristics are estimated based on hydrodynamic theory with account of elastic deformations occurring in vessel walls. Calculated values of dynamic blood pressure and flow velocity are compared with those of physiological norm, pathological states (aplastic anemia and hypertension). The possibility of blood flow monitoring with help of model developed is discussed.

Key words: blood flow, adaptation, arterial pressure, pulse wave, vessel dilatation, hydrodynamic model

Introduction

As it was noted earlier, for example in [1-3], the phenomenon of self-regulation was observed at blood supply. The phenomenon consists in ensuring physiologically optimum minute volume of blood under considerable variations in demand of the main organs. Such a situation is because of existing hierarchy within systems and subsystems of the organism having regulation elements at each level, organ or tissue, when every individual arteriole has its own regulation system. One of the major roles in self-regulation mechanism of blood flow belongs to baroreceptors of vessels. For example, raising of arterial pressure brings about tension of aorta walls, while pulsation of receptors leads to reduction of muscle tonus and vessel dilatation.

In the previous study [4], a mechanical problem of boundary localisation has been formulated for blood flow simulation taking into account the blood pressure, vessel walls elasticity and active deformation of vessels. Inner contour (vessel lumen) was considered to be the moving boundary, while optimum criterion was the minimum deviation of blood flow volume from the required level of blood supply.

For the estimation of dynamic blood pressure factor with linear approximation of passive (elastic) reaction of vessel walls the aim of present study was to obtain a numerical method of determining pulse wave parameters.

Deformation behaviour of cardiovascular tissues

Morphological investigations show that vessel walls usually consist of several layers with different mechanical properties [2, 5]. Their inner layer includes elastin covered from inside by smooth cells. The middle layer is formed by smooth muscle fibres whose degree of development varies in different vessels reducing with their diameters. The outer layer is formed from the most tough collagen fibres with elastin addition. Therefore a considerable anisotropy of mechanical properties is observed which has been studied elsewhere [4].

So, the existence of the developed muscle layer determines to a great extent physical nonlinearity of vessel walls. For example, 100 μm in diameter arterioles have an active layer consisting of three rows of muscle cells with about 5 μm thickness. In terms of adaptive systems, smooth musculature plays the role of actuating phase of the material. Moreover, the presence of baroreceptors in vessel is a prototype of self-informative material [6]. Mechanical testing of arterioles is, however, highly complicated and it is possible to obtain only indirect information about their mechanical properties based on physiological investigations.

When characterising deformation behaviour of cardiovascular tissues as a whole, it should be born in mind that there is not a single natural state to which elastic medium returns upon outer load removal. An analogue to it can be a reproducible state of either mechanical or chemical equilibrium. It means that non-invasive tests or preconditioning samples are necessary before studying mechanical properties of these biomaterials.

As far as heterogeneity, anisotropy and physical non-linearity of biomaterials necessitate complex theoretical descriptions, it is worthwhile making some simplifications as applied to effective solution of the partial hydrodynamic problem. These simplifications are relevant if we bear in mind the comparative analysis of pulse wave propagation for the cases of physiological norm and pathology (or surgical invasion).

Since most of biotissues show very low relaxation time limit $\tau = 10^{-8}$ s, so their deformations can be assumed reversible (although with noticeable hysteresis) and σ–ε dependence is almost linear for tension stress $\sigma > 200$ g/cm^2 [7]. Elasticity module of wall material can be assumed not to depend upon deformation rate that also simplifies the investigation.

Taking into account these remarks, the hypotheses of isotropy, incompressibility and linear elasticity of vascular tissue can be accepted for numerical modelling.

Hydrodynamic aspect

Dynamic phenomena in the blood circulation system are conditioned by blood flow pulsation at systole. Into circulation system of adults blood is forced in amount of 4-5 l/min in the state of rest at 100 torr pressure. Each systole supplies about 70 ml of blood into aorta and other coarse arteries.

Pressure pulses observed at heart work quickly propagate (in comparison to flow rate) along vessel walls and are the precursors of blood pressure increment [5]. These pulses are sensored by baroreceptors which initiate a purposeful muscle reaction of the vessels, namely, their dilatation, i.e. widening of their inner diameter.

The majority of works on pulse wave propagation are based on linearised theories. Since mean blood flow rate (about 25 cm/s in aorta) is small in contrast to pulse wave propagation (500 to 1200 cm/s), nonlinear members in equations for liquid motion are neglected. Blood viscosity is usually taken constant, although in different vessels it differs tens times and at low shear velocities blood behaves like a non-Newtonian liquid. To study pulse wave propagation and behaviour of arteries at various dynamic effects a hypothesis of liquid flow along a rectilinear section of vessel like an elastic shell is usually used. More accurate results can be obtained based on a model of a bent elastic pipeline and asymptotic solutions [8].

Calculation of flow parameters

The main parameters of blood flow in local parts of the circulation system are calculated by formulae which are given in [2].

Pressure losses (torr or mmHg) were calculated by formula which follows from Poiseuille's law for the laminar liquid flow

$$\Delta p = 336\frac{vq}{d^4}l, \qquad (1)$$

where *v* is the kinematic viscosity of blood, sSt; *q* is the volume of blood flow per second, *d* is the vessel diameter, mm; *l* is the vessel length, cm.

Owing to the non-linear dependence (1), dilatation of vessels leads to perceptible increase in carrying capacity**.**

The value of pulse wave front at the initial section of vessel and its variation depending on distance were calculated by Zhukovsky's formula

$$\Delta p_{\phi} = \frac{r \cdot w \cdot c}{1330}, \qquad (2)$$

where Δp_{ϕ} is the dynamic component of pressure on wave front; *r* is the blood density; *w* is the linear velocity of blood flow; *c* is the propagation rate of pulse wave.

Wave front losses at *l* distance from the source is equal to

$$\Delta p_{\Gamma} = e^{-0.7l\frac{n}{c \cdot 60}}, \qquad (3)$$

where $\frac{n}{c \cdot 60}$ is the wave length.

Change of vessel diameter in response to inner pressure is calculated by the formula following from Poiseuille's relation

$$d_p = d_0 / \sqrt{1 + \frac{1330 \cdot \Delta p}{r \cdot c^2}}, \qquad (4)$$

where d_0 is the initial vessel diameter before dilatation.

Minimum blood pressure was estimated from condition

$$p_{min} - \sum \Delta p \geq p_0, \qquad (5)$$

where ρ_0 = 5 torrs is the pressure at the capillary end; $\sum \Delta p$ - pressure losses within heart to capillary end distance.

Blood volume per second in a vessel is determined by the formula

$$q = \frac{V_s \cdot n}{60 \cdot k}, \qquad (6)$$

where V_s is the systole volume; *n* is the pulse frequency; *k* is total number of used vessels of a given diameter.

Linear velocity is determined from relation (1)

$$w = 2123\frac{q}{d^2}, \qquad (7)$$

where *q* is the blood volume per second; *d* is the vessel diameter.

Numerical results

Developed in DOS system computer model makes provision for the analysis of blood flow in real time. The following initial data were preset for calculations with assumption of laminar flow in the blood circulation system.

Table 1. Characteristics of blood vessels [2].

Diameter	Length	Number	Coefficient of activation	Coefficient of dilatation
10	40	1	1	$\left(1-\frac{1-k}{2}\right)$
3	20	40	1	$\left(1-\frac{1-k}{2}\right)$
1	10	600	1	k
0.6	1	1800	1	k
0.02	0.35	$4{\cdot}10^7$	1	k
0.007	0.09	$4{\cdot}10^8$	n_3	k
0.004	0.02	$18{\cdot}10^8$	n_3	1

Blood density and viscosity were taken 1.08 g/cm^3 and 14 sSt, respectively, according to data from [2]. Relative number of activated arterioles and capillaries was determined from the relation of current n and maximum $n_{\max}$ pulse frequency $n_3 = n/n_{\max} = 0.25$. The ratio of the systole time to actual one has been defined as $n_1/n_2 = 0.25$. Geometrical characteristics of blood channel, including calculated coefficient k of vessel dilatation/constriction relative to the norm which determines the actual vessel diameter are given in Table 1.

The proposed hydrodynamic model has been used to calculate blood circulation volume per minute, minimum and maximum blood pressures, vessel diameter and blood flow rate through a given section. The initial parameters were pulse frequency, systole volume, blood density and viscosity, velocity of pulse wave propagation and coefficient of vessel dilatation. Modelling results make it possible to trace variations in pressure, flow rate and vessel diameter in time or distribution of named parameters along vessel length in a certain moment of time.

Two groups of data can serve as the initial for developed HDM (hemodynamic model) computer program. Firstly, these include information obtained at patient monitoring in clinic, namely pulse frequency blood volume at systole, the relation between systole and diastole periods, blood viscosity and density and sound velocity in the blood circulation system. The second group includes expected values of maximum pulse frequency and those of constriction/expansion of vessels. The data make it possible to draw diagrams of blood flow velocity, arterial pressure and vessel diameter in a certain time period for vessels of a given type.

At present linear approximation of cardiac cycle is used based on systole to diastole time ratio and required pressure maximum to allow blood into capillaries.

Based on known characteristics of blood flow at normal and preuremic states, and aplastic anemia, the parameters of necessary changes in diameters at pressure minimum and maximum in case of norm, expansion coefficients for aorta and arteriole diameters can be given as an example (Table 2).

Calculated values of pressure and blood flow rate in two kinds of vessels (aorta and arteriole) were compared for the states of physiological norm, aplastic anemia and hypertension (Fig. 1). As it is seen from Fig. 1, blood pressure reaches its maximum at a certain moment (distance from pressure source).

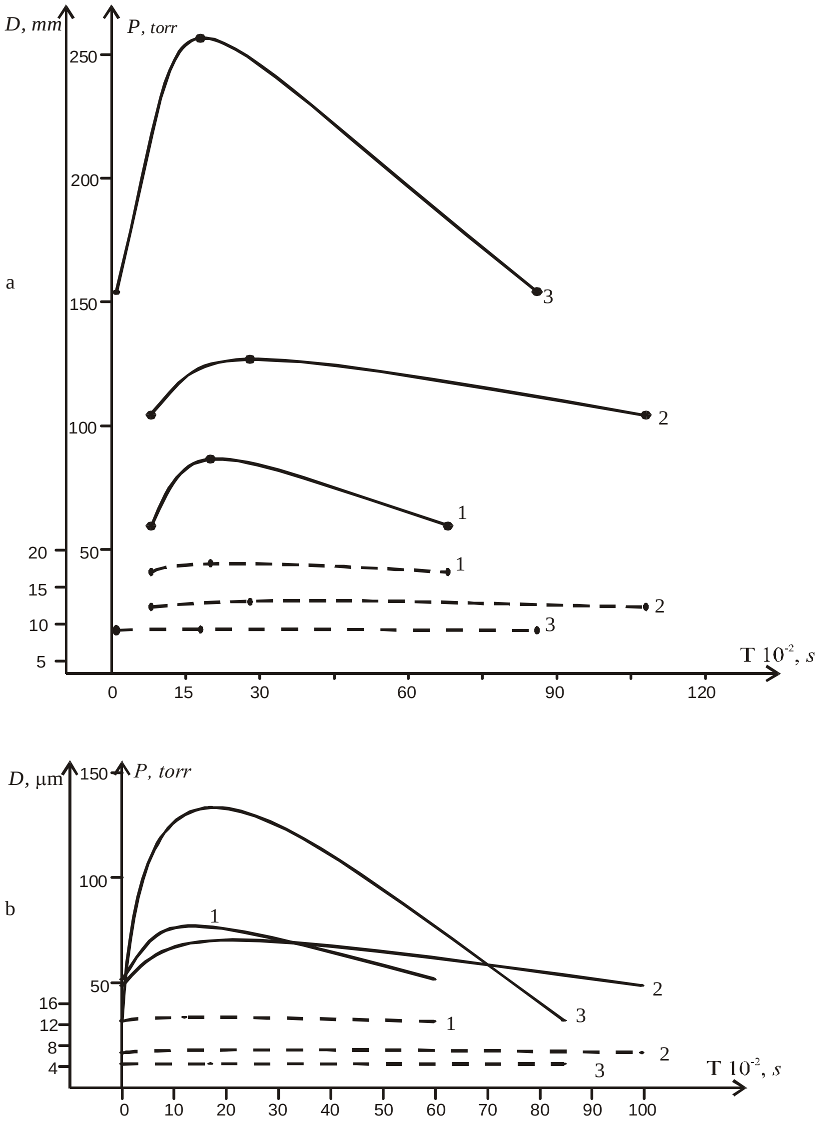

Fig. 1. The distributions of pressure p and vessel lumen D for aorta (a) и arteriole (b):
1 – preuremic state; 2 – normal state; 3 – aplastic anemia.

Table 2. Comparison of different physiological states parameters.

Patient state	Literature data						Modeling results						
								Aorta			Arteriole		
	pulse frequency	max pulse frequency	systole volume	sound velocity	min pressure	max pressure	required coefficient	diameter variation ratio	min pressure	max pressure	diameter variation ratio	min pressure	max pressure
Aplastic anemia	100	180	0.11	500	45	90	2.0	1.07	59	86	1.06	52	77
Healthy	60	180	0.07	500	100	120	1.0	1.06	104	127	1.05	49	70
Preuremic state	70	180	0.03	2400	145	250	0.8	1.01	154	256	1.01	32	133

To promote the computer analysis it is necessary to exclude coefficient of standard diameter variation (obtained now by probe) and, probably, sound velocity from input data. This will assist in constructing identification models of the state of peripheral blood system since sound velocity describes vessel tension, while coefficient of diameter variation at normal state reflects degree of blood channel deformation.

Of a paramount interest is adding to the model of a “thrust-free reservoir” for discharge in zone of thrust creation of valve function. This can be useful in modelling valvular diseases which are the reason of low efficiency of heart work in the phase of diastole expansion.

Conclusion

The proposed hydrodynamic model of arterial system has demonstrated experimentally observed profiles of blood pressure in norm and pathology. This model may be used as the element of computer monitoring of blood flow in real time.

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РАСЧЕТ ХАРАКТЕРИСТИК ПУЛЬСОВОЙ ВОЛНЫ С УЧЕТОМ ДЕФОРМАЦИЙ КРОВЕНОСНЫХ СОСУДОВ

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Анализируется механизм авторегуляции артериального кровотока, обеспечивающий оптимальное функционирование сердечно-сосудистой системы. Определение характеристик пульсовой волны в артериях производится на основе гидродинамической теории с учетом деформаций стенок сосуда. Сопоставляются расчетные значения давления и скорости кровотока для физиологической нормы, апластической анемии и гипертензии. Обсуждается возможность использования модели для диагностики параметров кровообращения. Библ. 8.

Ключевые слова: кровообращение, адаптация, артериальное давление, пульсовая волна, дилатация сосуда, динамическая модель

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